two patients lived over two years in perfect well-being, the other three years, before showing recurrence. It is a well-known fact that recurrence in cancer of the stomach is quite often delayed. While the small percentage of permanent cures is very discouraging, a large number of patients enjoy at least two years of perfect well-being before getting recurrence after partial gastrectomy.

The fact that even incomplete resection sometimes gives a fair result should not, of course, tempt the surgeon to resect too close to the tumor. Accelerated growth and rapid recurrence can often be expected to follow. After cutting across the stomach, I have lately been excising an additional strip of tissue from the proximal stump for immediate frozen section. If the pathologist reports presence of cancer cells in this strip, more of the stomach is resected, if technically possible.

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THOMAS O. BURGER, M. D. (1301 Medico-Dental Building, San Diego).—Cancer of the stomach is curable by only one method—surgery, and that surgery must be complete eradication of the growth and gland-bearing areas. Surgery, therefore, must be early.

Cancer in almost any location of the body is not *painful,* and that is true of cancer of the stomach, as shown in Doctor Woolsey's charts.

Cancer of the stomach untreated is fatal, and has a very distressing final stage. Cancer of the liver is not a painful illness. Surgery should be radically done with attempt to cure, even though the mortality rate is rather high in extensive resections. If the attempt at complete eradication is not successful, but the patient recovers from the surgery, the recurrence is usually much less terrible in its pain and starvation in the later stages than when no surgery is done. This is true, as already said, if the liver is the location of recurrence, as is often the case.

In some patients, high resection is very difficult, and we find that, especially in high resections, the De Petz sewing instrument makes not only easier work, but will cut the time of operation about twenty minutes. Twenty minutes may mean saving a life in a good number of these persons.

The x-ray is the principal agent in proving or disproving a stomach trouble in regard to cancer, and should always be resorted to in persons of forty years or over. Also a recheck should be made if any question as to whether there is an ulcer or cancer.

Early diagnosis and radical surgery means a large per cent of cures; and if not cures, a prolongation of comfortable living for a considerable period of time.

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JOHN HUNT SHEPHARD, M. D. (Medico-Dental Building, San Jose).—In analyzing Doctor Woolsey's records, Table 11, showing that only twenty of the ninety-seven cases coming to operation were resectable, closely agrees with other published reports. Viewed in the light of Table 4, showing the length of time these patients had been under medical care prior to operation, Table 11 places the profession directly "on the spot."

Our efforts in educating the laity concerning cancer has accomplished a great deal, and already our experience leads us to believe that the individual who feels a tumor in the breast, sees evidence of a lesion on the skin or lip, or develops a vaginal discharge in lesions of the cervix, not only seeks early advice, but when expectant or temporizing treatment is advised immediately consults another physician.

We must not expect the laity to be their own diagnosticians and tell us what to do. The symptoms of gastric disturbance should be as readily read by the physician as the suggestive signs of cancer of the breast or cervix are read by the patient, and the importance and necessity of a proper gastric examination recognized. While occasionally a patient whose symptoms have persisted for only a short time will refuse to submit to a complete examination, our experience leads us to believe that the physician is not infrequently responsible for the delay in making a thorough examination, either through his own suggestion, or by his ready consent to try this or that form of treatment, trusting that nature will effect a cure. It may be that this willingness to "wait and see" results from the physician's desire to curtail expenses to his patient; but since the same practice is found in charity clinics, I believe it is due to the lack of proper appreciation on the part of the doctor, of the importance of a thorough and complete study of every symptom of which the patient complains.

For years, every patient over forty years of age, consulting us on account of stomach trouble which has existed for more than one week, has been subjected to an x-ray examination before instituting treatment. Of those who have not complied with our rule, some have been cured by nature, some have had their examination made elsewhere, and some have returned with an inoperable carcinoma of the stomach.

When we of the medical profession cease to allow our patients to direct the course of our study, and insist on a thorough examination before instituting treatment, carcinoma of the stomach, as well as other maladies, will be recognized at a time when treatment may effect a cure.

ROENTGENOLOGIC CHANGES IN MALACIC DISEASE OF BONE*

By JOHN D. CAMP, M. D.
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THE present interest of the medical profession in the subject of malacic disease of bone was stimulated largely by the report of Mandl,[1] in 1926, concerning a case of osteitis fibrosa cystica generalisata (von Recklinghausen's disease), in which the removal of a parathyroid adenoma was followed by prompt clinical improvement. The literature concerning the subject from that time has become voluminous, and the existence of "hyperparathyroidism" as a definite clinical entity has become well established. Because of the profound influence of the parathyroid glands on the fundamental metabolism of calcium, and probably of phosphorus, it is not surprising that the question of parathyroid function, as related to other forms of malacic disease of bones of supposedly known and unknown origin, has been seriously considered. Some enthusiastic investigators of the subject see in the condition of hyperparathyroidism a ready explanation for the cause of such conditions as osteitis deformans (Paget's disease), leontiasis ossium, ankylosing polyarthritis of Oppel, giant-cell tumor, multiple myeloma, and scleroderma. Other observers, whose conclusions concerning the subject are tempered by experimental evidence, are less ready to accept such a broad application of the term "hyperparathyroidism." Because of such conflicting opinions no little confusion has resulted, particularly in the minds of roentgenologists, concerning the identity of certain forms of malacic disease of bone. Without doubt the present intensive clinical and investigative work that is being carried out on the subject of hyperparathyroidism and mineral metabolism in general will

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TABLE 1.—*Classification of Malacic Disease of Bone*

Group 1.—Atrophy associated with:	
Disuse	Neurotrophic disorders
Fractures	Circulatory disorders
Arthritis	
Group 2.—Congenital defects	
Osteogenesis imperfecta	
Fragilitas ossium	
Group 3.—Dietary insufficiency and avitaminosis	
Rickets	
Osteomalacia	
Puerperal	Celiac disease
Senile	Chronic idiopathic steatorrhea
Hunger and war deficiency	Kashin-Beck disease
Group 4.—Renal insufficiency	
Renal rickets	
Group 5.—Endocrine malfunction	
Hyperparathyroidism	Suprarenal disease
Hyperthyroidism	Diabetes mellitus
Cushing's syndrome (Basophilism)	
Group 6.—Miscellaneous: Etiology unknown	
Focal osteitis fibrosa cystica	
Osteitis deformans (Paget's disease)	

have a far-reaching effect on our knowledge and classification of certain forms of lesions of bone. With a full realization of this situation, the classification in Table 1 is offered, subject to revision. It does offer to my mind, at the present time, however, a reasonable basis for consideration of the roentgenologic changes in the various forms of malacic diseases of bone.

Because of the multiplicity of conditions that may affect the mineral content and structure of the skeleton, the early recognition of any particular disease by roentgenograms alone may be exceedingly difficult. Even in some advanced lesions, as will be pointed out subsequently, the ultimate diagnosis can be made only by proper correlation of the clinical data and other laboratory findings.

GROUP 1. ATROPHY

Atrophy of bone resulting from disuse incident to trauma, fracture or arthritis, neurotrophic disorder, or to circulatory disease, is characterized by its limitation to the affected part. Two types may be observed: (1) a homogeneous decalcification of bone with thinning of the trabeculae, which, however, remain sharply defined despite their thinness; and (2) a coarse, mottled form of osteoporosis, which has been confounded with multiple myeloma and with metastatic malignancy. This type has been called subacute atrophy of bone; but this is a misnomer, since it may occur in such chronic states as Buerger's disease, arteriosclerosis, and in peripheral nerve injuries (Fig. 1).

GROUP 2. CONGENITAL DEFECTS

In this group are placed those cases in which the bones are fragile, not because of any primary defect in calcium metabolism, but because of a primary defect in the mesoblast, as a result of which connective tissues are unable to build up a sufficient framework on which enough lime may be deposited. In this class are placed osteogenesis imperfecta and fragilitas ossium, probably the same pathologic process, except that the former concerns the fetal or infantile form and the latter the adult. Both have a tendency to be hereditary and familial. The entire skeleton is affected, and roentgenologic changes in the infantile form are characteristic. The bones have a bowed, misshapen, shortened, and thickened appearance. Callus is easily detected in the shafts at the sites of fracture. Compression of the thorax and fracture of the ribs are common. Ossification in the skull is deficient and irregular and, according to Knaggs,[2] has a peculiar mosaic appearance due to the irregular islands of bone occurring particularly along suture lines. In the adult variety, there is generalized osteoporosis and marked atrophy of the cortex and shafts of the long bones. Bowing, infractions, and fractures occur in varying degrees.

GROUP 3. DIETARY INSUFFICIENCY AND AVITAMINOSIS

Rickets and osteomalacia constitute the lesions of this group, which result from a deficient supply of calcium or phosphorus in the food or a deficient intake of vitamin D. There is little doubt that they are one and the same disease, except that rickets affects the growing child and osteomalacia the adult. The roentgenologic changes of rickets are so well known that they will not be reviewed here. In the past the term "osteomalacia" has, by tradition, been applied to the particular softening of bones occurring in pregnant and lactating women. Experience has shown, however, that such changes are not limited to pregnancy and lactation, but may occur whenever the diet is deficient in calcium, phosphorus, or in vitamin D. Notable examples of this include hunger and war deficiency, and the changes in bone occurring in celiac disease and in chronic idiopathic steatorrhea. The significant roentgenologic changes consist of generalized osteoporosis, marked thinning of the cortical bone, with softening, bowing, fractures, and generalized deformities. Of particular interest are the transverse osteoid zones, described by Loöser,[3] which simulate pathologic fractures (Fig. 2). They usually occur in well-advanced cases, and have a tendency to be symmetrically distributed. Osteomalacia is frequently confounded roentgenologically with the skeletal changes in hyperparathyroidism, from which it differs in that osteoporosis is homogeneous or coarsely striated rather than granular in character, and that cysts do not form.

GROUP 4. RENAL INSUFFICIENCY

Renal rickets is a condition developing in children in association with, and apparently as a result of, chronic nephritis. Roentgenologically, the findings vary a great deal in different cases, and sometimes in the same case at various times. Skeletal development is always retarded. In some instances there is a zone of osteoid tissue at the growing ends of bones which may not be different from that seen in ordinary rickets. However, there usually is something atypical about the findings, and the lesions may be asymmetrical, or different bones may be unequally involved. In

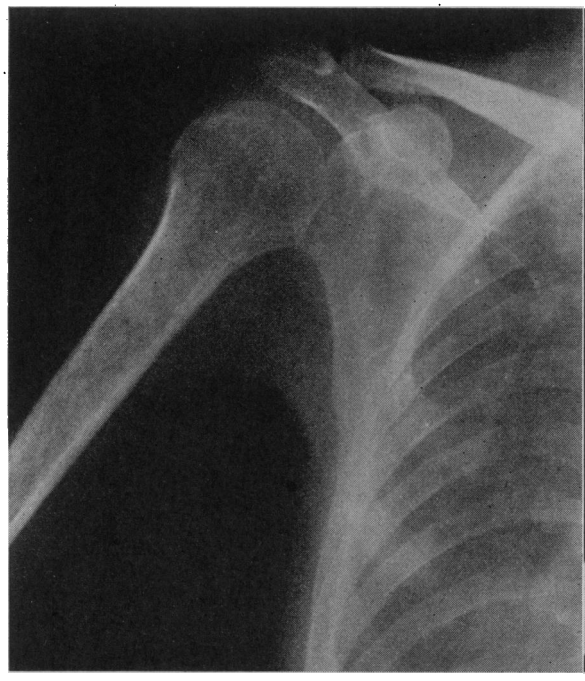

Fig. 1.—Coarse, mottled type of osteoporosis of humerus, resulting from old injury to brachial plexus.

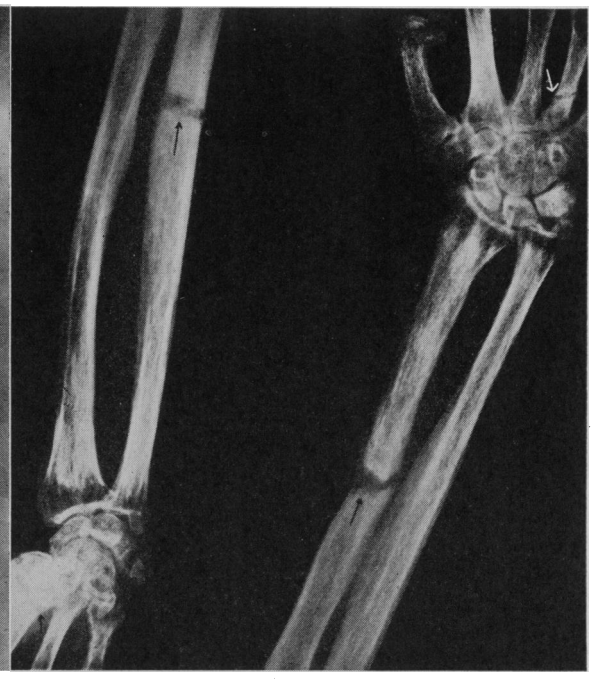

Fig. 2.—Osteomalacia, resulting from dietary insufficiency and deprivation of sunlight. The osteoid zones which simulate fractures may be noted.

cases of long standing, there is a deep mottled zone at the ends of long bones which consists of osteoid tissue, cartilage, fibrous tissue, and calcified matrix. Distortion, infractions, and various deformities may result. Defects in subperiosteal calcification and a coarse, thickened appearance of the skull in advanced cases have been noted by Parsons [4] and Teall.[5]

GROUP 5. ENDOCRINE MALFUNCTION

Hyperparathyroidism.—This is a disease which is usually, if not always, due to a functioning adenoma of the parathyroid glands. As a result of increased production of hormone, there is disturbance in the metabolism of calcium and phosphorus, which results in certain generalized structural changes in the skeletal system, the roentgenographic appearances of which are characteristic. Roentgenologically, the fundamental skeletal change (demineralization) is revealed in the majority of cases as a uniform, miliary, granular type of osteoporosis. This peculiar form of generalized, mottled atrophy is distinct from the ordinary type seen in osteoporosis associated with acute and chronic disease of bone and in neurotrophic conditions. It is best exhibited in flat bones, especially in the calvarium (Fig. 3). Also, resorption causes trabeculae of bones to become indistinct in outline and cortical bone to be so thinned that, in early cases, the bones have a homogeneous ground-glass appearance. In some regions decalcification progresses to produce multiple cystic zones (osteitis fibrocystica generalisata) of varying size, which may be found within the medullary portion or below the periosteum. The jaws, pelvis, long bones, ribs, and metatarsal and metacarpal bones are favorite sites for such changes. The subperiosteal zones of resorption are especially well shown at the ends of the bones and in the phalanges. Cysts may reach a large size and become the site of pathologic fracture. Because the bones are soft, bowing, kyphosis, narrowing of the pelvis, and coxa vara are common. The onset of the disease is so insidious that, in early cases, it is difficult to distinguish it from osteoporosis resulting from other causes.

Following the removal of a parathyroid tumor, a very obvious change occurs in the structure of the skeletal system. As the resorption of calcium is arrested, the trabeculae of bones lose their indistinct outline and become well defined. As convalescence occurs, the density of the bone gradually approaches the normal, although such

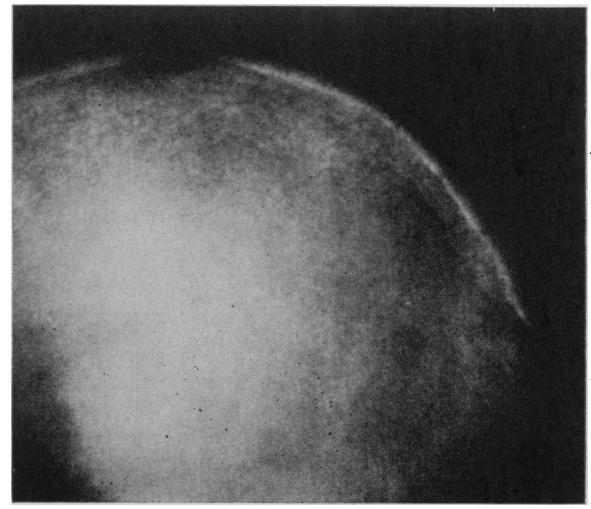

Fig. 3.—Section of roentgenogram of skull revealing characteristic granular osteoporosis of hyperparathyroidism.

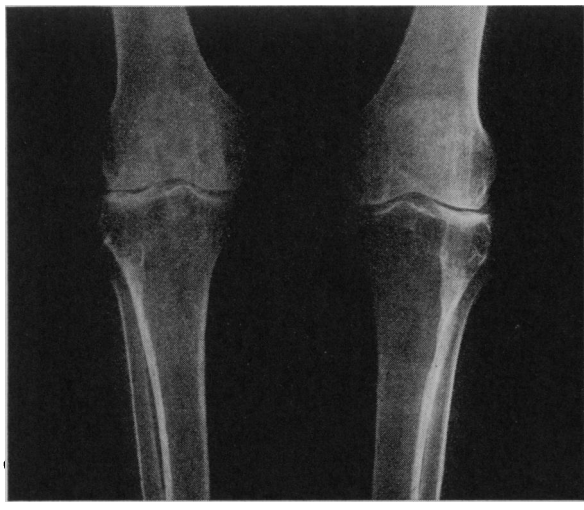

Fig. 4.—Osteoporosis associated with exophthalmic goiter.

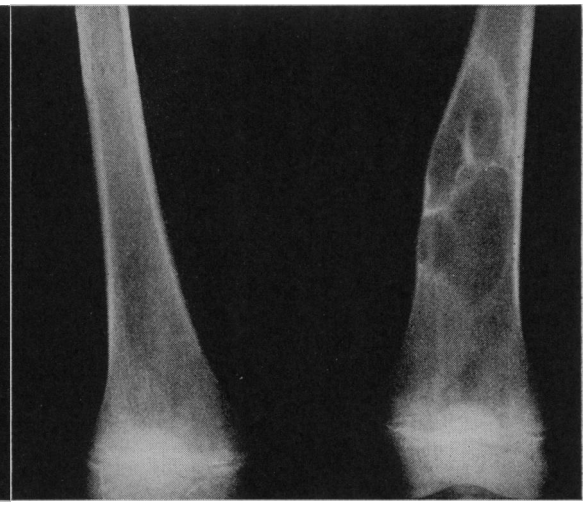

Fig. 5.—Focal osteitis fibrosa cystica. The presence of normal bone in the uninvolved areas distinguishes this condition from hyperparathyroidism.

deformities as bowing, kyphosis, and scoliosis will persist. Cystic regions gradually fill in and may disappear completely.

Hyperthyroidism.—The presence of osteoporosis in exophthalmic goiter was demonstrated radiographically by Kummer[6] in 1917. Since that time several observers have confirmed his observations. The demineralization is generalized and homogeneous in character, but is more marked in the spongy portions (Fig. 4). In one of a series of cases reported by Plummer and Dunlap[7] in 1928, necropsy revealed generalized osteoporosis, especially of the calvarium and ribs, the latter disclosing multiple fractures. According to Hunter,[8] there seems to be no relation between the duration of the disease, the degree of osteoporosis, and the values for phosphatase in the plasma, which often are high.

Cushing's Disease (Basophilic Adenoma of the Pituitary Gland).—Osteoporosis has been one of the features of a syndrome associated with basophilic tumors of the anterior lobe of the pituitary gland. In the few cases that I have seen, osteoporosis has been of the generalized homogeneous type and would not have been confused with the granular osteoporosis of primary hyperparathyroidism. Whether or not secondary hyperparathyroidism is present, due to hyperplasia of the parathyroid gland, has not been definitely established. The obesity, hirsutism, amenorrhea, and hypertension in Cushing's syndrome certainly are not present in primary hyperparathyroidism, and they should thus serve to distinguish the condition clinically if the roentgenographic changes are inclusive.

Suprarenal Disease. — The literature contains much conflicting data regarding the relation of the suprarenal glands to calcium metabolism and to osteomalacia, and the present status of our knowledge concerning this matter is well summarized by Golden and Abbott,[9] who concluded that, although suprarenal secretion either directly or indirectly influences calcium metabolism, and although the suprarenal glands may be indirectly involved in a pluriglandular imbalance in certain cases of osteomalacia, the evidence did not seem to justify the assumption that decalcification of bones results directly from suprarenal disease or dysfunction.

Diabetes Mellitus.—There is considerable evidence in the literature that a loss of calcium occurs in diabetes, especially in the presence of acidosis. Morrison and Bogan[9] found that atrophy of bone occurs among a certain number of children in whom diabetes develops before the ninth year, but not after that. Narrowness of the shaft and thinness of the cortex were noted in the bones of children with long-standing diabetes. Decalcification was observed in diabetes among adults by Golden and Abbott[10]; but according to their experience the question may be raised as to how much of this is of endocrine and how much of nutritional origin. They concluded that roentgenologic evidence of definite, important, skeletal decalcification among diabetic adults, which can be attributed directly to the disease, is lacking.

GROUP 6. MISCELLANEOUS—ETIOLOGY UNKNOWN

Focal Osteitis Fibrosa Cystica.—Localized or focal osteitis fibrosa cystica has been termed by von Mikulicz,[11] "osteodystrophia cystica juvenilis." This condition is frequently confounded with hyperparathyroidism because, roentgenologically and pathologically, the localized cysts simulate the cystic lesions of hyperparathyroidism. The condition is not related to hyperparathyroidism, and can be distinguished from it by the presence of normal bone in the uninvolved region (Fig. 5), and by the presence of normal amounts of calcium and phosphorus in the serum, and of phosphatase in the plasma.

Osteitis Deformans (Paget's Disease). — The roentgenologic characteristics of this disease, which may affect one or several bones, are so well known

that they need not be repeated here. Of more importance is the question whether or not this disease is a manifestation of hyperparathyroidism, as is claimed by some observers. By analogy with generalized osteitis fibrosa cystica, it would seem likely that Paget's disease is a disorder of mineral metabolism and, according to Turnbull,[12] there is only one definite histologic difference between the two diseases; this, however, is a big difference. In osteitis deformans, there is tremendous resorption of bone associated with tremendous apposition which completely alters the normal architecture, although apposition predominates. In generalized osteitis fibrosa cystica, widespread resorption of bone is the essential change, and excessive apposition is focal and relatively insignificant. Enlargement of the parathyroid glands has not been demonstrated in Paget's disease, and the levels of calcium and phosphorus in the serum are normal in contrast to the levels in hyperparathyroidism.

In addition to the conditions that have already been discussed, there occur certain other diseases which, because of their involvement of the osseous system, may be confounded roentgenologically with true malacic diseases of bones. These include metastatic carcinoma, multiple myeloma, diseases of the reticulo-endothelial system, and certain blood dyscrasias, particularly those of infancy and childhood.

The extensive involvement of the skeletal system in advanced cases of metastatic carcinoma and multiple myeloma may at first glance simulate the roentgenographic appearance of hyperparathyroidism. However, the multiple regions of destruction of bone in these conditions, in the calvarium especially, are easily distinguished from the miliary, granular, osteoporotic lesions in the skull in hyperparathyroid disease. True formation of cysts is absent in each of the above conditions, whereas it is common in hyperparathyroidism. In hyperparathyroidism the entire skeleton is affected, whereas in multiple myeloma and metastatic carcinoma zones of uninvolved normal bone can generally be found regardless of the extent of the disease. Because of marked skeletal destruction, determinations of calcium and phosphatase may be high. However, serum phosphorus, if changed, is higher than normal in contrast with the low serum phosphorus in hyperparathyroidism, except in the presence of severe renal injury when phosphorus in the serum may be elevated. The fact that hyperplasia of the parathyroid glands may occur in myeloma has been cited as evidence that this disease is of parathyroid origin. However, hyperplasia of the parathyroid glands has been observed also in metastatic carcinoma of bone, in rickets, and in osteomalacia, and the majority of observers agree that it is undoubtedly a secondary rather than a primary factor in these conditions. Also, reliable records exist of cases of multiple myeloma wherein the calcium and phosphorus metabolism was normal, and normal parathyroid glands were found at necropsy.

Disease of the Reticulo-Endothelial System.— Xanthomatosis (Schüller-Christian syndrome), according to Rowland,[13] is a rare, probably familial, constitutional disorder of metabolism in which a deposition of lipoid mixtures, particularly cholesterol and its esters, takes place, leading to characteristic hyperplastic reaction in the reticuloendothelial or histiocytic apparatus. It occurs most frequently in early childhood, but occasional adolescent and adult types have been noted. The resorption of bone is really a destructive process or pressure atrophy caused by contiguous accumulation of the granuloma-like lipoid masses in the connective tissue. The flat bones are more commonly involved than other bones, but any part of the skeleton may be affected. The plaque-like zones of destruction in the skull are quite different from the granular osteoporosis of hyperparathyroidism, also the unaffected bones and uninvolved regions of the same bone exhibit a normal structure in contrast to the diffuse involvement in hyperparathyroidism.

Gaucher's Disease. — This condition provokes changes in the skeletal system that are more similar to osteomalacia than to hyperparathyroidism. The crowding of bone marrow with so-called Gaucher cells results in atrophy of the spongy portions of the bones and widening of the intramedullary space. While the changes must be generalized, they are particularly obvious in the ends of the femur and humerus. According to Schinz,[14] change in the skull is observed only in advanced cases. Because of the hematologic findings and the presence of splenomegaly, this condition would not be confused clinically with osteomalacia.

Blood Dyscrasias. — The roentgenologic manifestations of these conditions have been described in detail by Karshner,[15] and by Diamond and Vogt. The latter investigators classify the whole group under the general heading "congenital anemias." Fundamentally, the changes in the osseous system are the result of a generalized hyperplastic condition of the marrow, which causes decalcification of the trabeculae and thinning of cortical bone. The changes are well shown in the calvarium where a generalized mottling or spongy appearance may be observed.

In the erythroblastic form, the medullary portion of the calvarium may be thickened, and the outer and inner tables correspondingly thinner. In a later stage of this disease, formation of new bone occurs, which is revealed as striations perpendicular to the tables. According to Karshner these striations in the skull suggest replacement of exhausted marrow by bone.

Among patients with leukemia, especially of the lymphatic variety, periosteal elevation and thickening due to subperiosteal leukemic infiltration, commonly occur. In addition, small or extensive foci of destruction of bone, which cause a moth-eaten appearance of the medulla, may be noted.

Clinically, the diagnosis of various congenital anemias is not difficult, although roentgenologically they may be confounded at certain stages with hyperparathyroidism and with osteogenesis imperfecta.

COMMENT

In reviewing the malacic diseases of bone from the standpoint of the roentgenologist, one cannot help but be impressed with the variety of roentgenographic changes that are presented when such a fundamental metabolic change as demineralization is provoked by various causes. Histologic studies offer a reasonable explanation for these variations and, in my opinion, a carefully correlated study of the microscopic changes and roentgenograms in the conditions noted in this paper will do much to further our knowledge of malacic diseases of bone, and of osteoporosis in general.

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REACTION FOLLOWING BLOOD TRANSFUSION*

REPORT OF AN UNUSUAL CASE

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AND
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San Francisco

DISCUSSION *by Roy W. Hammack, M. D., Los Angeles; LeRoy Brooks, M. D., San Francisco.*

CASE HISTORY (By J. O. Haman). Mrs. R., thirty-two, was first seen in the Prenatal Clinic in November, 1932, five months pregnant.

The family history and past history were negative, except for typhoid and pneumonia. Catamenia began at seventeen, was always irregular, painful and profuse.

There had been four previous pregnancies, the first in 1927. This progressed normally until one week before labor was due, when headache, diarrhea, vomiting, edema, high blood pressure and albuminuria developed. She was taken to a hospital semi-comatose and in labor; the pregnancy was terminated with high forceps. She had several convulsions after delivery, which were treated by venesection. Later she was given a blood transfusion, her husband being the donor. A mild reaction, consisting of chills and fever, developed immediately, but soon subsided. Blood pressure and urine were normal after the puerperium.

The second and third pregnancies (1928 and 1930) aborted spontaneously at four months, each time with toxic symptoms developing prior to the miscarriage. The fourth pregnancy (1931) was complicated by hypertension and albuminuria throughout. Three weeks before delivery the patient developed oliguria and was under hospital observation for twenty-two days, when she began hemorrhaging. Labor was induced by a Voorhees bag, and a podalic version and extraction were performed. Urine and blood pressure were again normal after this pregnancy.

The present pregnancy was normal throughout, except for a blood pressure which remained at approximately 145/85. Slight traces of albumin were noticed at intervals. Hemoglobin was 65 per cent Sahli. Other laboratory tests and physical examination were negative, except for marked laceration and erosion of the cervix. Twins were diagnosed at the eighth month. Treatment during pregnancy consisted of iron in the form of Blaud's pills, and a low-protein, salt-free diet.

The patient was delivered on March 28, 1933, after a labor of six and three-fourths hours. Fraternal twins were born, each weighing six and one-half pounds. The first was a normal, vertex presentation; the second was in a transverse position, and a podalic version and extraction were performed. The hemoglobin on entry was 62 per cent Sahli, the urine showed nothing abnormal, and the blood pressure was 146/88. As the patient appeared very pale, and her hemoglobin was only 50 per cent Sahli, a transfusion of 400 cubic centimeters citrated blood was given on the third day after delivery, her husband again being the donor. The laboratory report showed both donor and recipient to be Type II (Moss), with the donor's cells showing slight rouleaux formation with the patient's serum. The donor was considered to be suitable if the blood were given slowly. The blood was injected into the median basilic vein at a rate of 7 cubic centimeters per minute, the procedure consuming fifty-five minutes.

Toward the end of the transfusion the patient suffered a severe chill which lasted twenty minutes. The temperature rose abruptly to 40.2 degrees centigrade, dropped to 36.8 degrees centigrade within twenty-four

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